

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1635jxs

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * Welcome to STN International * * * * * * * * *

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America
NEWS 2 "Ask CAS" for self-help around the clock
NEWS 3 DEC 23 New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/
 USPAT2
NEWS 4 JAN 13 IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS 5 JAN 13 New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
 INPADOC
NEWS 6 JAN 17 Pre-1988 INPI data added to MARPAT
NEWS 7 JAN 17 IPC 8 in the WPI family of databases including WPIFV
NEWS 8 JAN 30 Saved answer limit increased
NEWS 9 FEB 21 STN AnaVist, Version 1.1, lets you share your STN AnaVist
 visualization results
NEWS 10 FEB 22 The IPC thesaurus added to additional patent databases on STN
NEWS 11 FEB 22 Updates in EPFULL; IPC 8 enhancements added
NEWS 12 FEB 27 New STN AnaVist pricing effective March 1, 2006
NEWS 13 FEB 28 MEDLINE/LMEDLINE reload improves functionality
NEWS 14 FEB 28 TOXCENTER reloaded with enhancements
NEWS 15 FEB 28 REGISTRY/ZREGISTRY enhanced with more experimental spectral
 property data
NEWS 16 MAR 01 INSPEC reloaded and enhanced
NEWS 17 MAR 03 Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 18 MAR 08 X.25 communication option no longer available after June 2006
NEWS 19 MAR 22 EMBASE is now updated on a daily basis
NEWS 20 APR 03 New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS 21 APR 03 Bibliographic data updates resume; new IPC 8 fields and IPC
 thesaurus added in PCTFULL
NEWS 22 APR 04 STN AnaVist \$500 visualization usage credit offered

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
 CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
 AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
 V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
 <http://download.cas.org/express/v8.0-Discover/>

NEWS HOURS STN Operating Hours Plus Help Desk Availability
NEWS LOGIN Welcome Banner and News Items

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 19:25:13 ON 10 APR 2006

FILE 'MEDLINE' ENTERED AT 19:25:22 ON 10 APR 2006

FILE 'BIOSIS' ENTERED AT 19:25:22 ON 10 APR 2006
Copyright (c) 2006 The Thomson Corporation

FILE 'CA' ENTERED AT 19:25:22 ON 10 APR 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'EMBASE'. ENTERED AT 19:25:22 ON 10 APR 2006
Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE 'SCISEARCH' ENTERED AT 19:25:22 ON 10 APR 2006
Copyright (c) 2006 The Thomson Corporation

=> f (u1 or u2 or snrna)
L1 25343 (U1 OR U2 OR SNRNUA)

=> s (u1 or u2 or snrna)
L2 25343 (U1 OR U2 OR SNRNA)

FILE 'MEDLINE' ENTERED AT 19:25:59 ON 10 APR 2006

FILE 'BIOSIS' ENTERED AT 19:25:59 ON 10 APR 2006
Copyright (c) 2006 The Thomson Corporation

FILE 'CA' ENTERED AT 19:25:59 ON 10 APR 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'EMBASE' ENTERED AT 19:25:59 ON 10 APR 2006
Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE 'SCISEARCH' ENTERED AT 19:25:59 ON 10 APR 2006
Copyright (c) 2006 The Thomson Corporation

=> s (u1 or u2 or snrna)
L3 25343 (U1 OR U2 OR SNRNA)

=> s antisense or anti-sense
L4 141392 ANTISENSE OR ANTI-SENSE

=> s 13 (2n) 14
L5 107 L3 (2N) L4

=> S 13 (P) 14
L6 443 L3 (P) L4

=> S 13 (S) 14
L7 290 L3 (S) L4

=> dup rem 17
PROCESSING COMPLETED FOR L7
L8 97 DUP REM L7 (193 DUPLICATES REMOVED)

=> s 18 and (pd<=1995)
1 FILES SEARCHED...
4 FILES SEARCHED...
L9 21 L8 AND (PD<=1995)

=> d 19 ibib abs 1-21

L9 ANSWER 1 OF 21 MEDLINE on STN
ACCESSION NUMBER: 95286698 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7768997
TITLE: The organization of spliceosomal components in the nuclei of higher plants.
AUTHOR: Beven A F; Simpson G G; Brown J W; Shaw P J
CORPORATE SOURCE: Department of Cell Biology, John Innes Centre, Colney, Norwich, UK.
SOURCE: Journal of cell science, (1995 Feb) Vol. 108 (Pt 2), pp. 509-18.
Journal code: 0052457. ISSN: 0021-9533.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199507
ENTRY DATE: Entered STN: 19950713
Last Updated on STN: 19950713
Entered Medline: 19950706
AB To analyze the organization of spliceosomal snRNPs in plant nuclei, we have used both immunofluorescence labelling with the antibody 4G3, raised against the human snRNP-specific protein U2B", and in situ hybridization with anti-sense probes to conserved regions of U1, U2 and U6 snRNAs. The organization comprises a fibrous interchromatin network, which may include both interchromatin fibrils and granules, and very prominent nuclear and nucleolar-associated bodies. Double labelling with an anti-p80 coilin antibody shows that these are coiled bodies. Dynamic changes in the labelling pattern were observed through the cell cycle, and in response to and on recovery from heat shock. The similarity of this organization to that observed in mammalian nuclei is strong evidence that it is fundamental to the processing of pre-mRNA in eucaryotes in general.

L9 ANSWER 2 OF 21 MEDLINE on STN
ACCESSION NUMBER: 95081156 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7989372
TITLE: Incorporation of 5-fluorouracil into U2 and U6 snRNA inhibits mRNA precursor splicing.
AUTHOR: Lenz H J; Manno D J; Danenberg K D; Danenberg P V
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Southern California School of Medicine, Los Angeles 90033.
CONTRACT NUMBER: AI07078 (NIAID)
SOURCE: The Journal of biological chemistry, (1994 Dec 16) Vol. 269, No. 50, pp. 31962-8.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199501
ENTRY DATE: Entered STN: 19950124

Last Updated on STN: 19950124
Entered Medline: 19950112

AB The splicing activities of 5-fluorouracil (FUra)-substituted U2 and U6 small nuclear RNAs (snRNAs) were examined in an in vitro splicing system. Yeast splicing extracts were specifically depleted of endogenous U2 and U6 snRNAs by antisense oligonucleotide-directed RNase H hydrolysis. Splicing activity was recovered when the extracts were reconstituted with synthetic U2 and U6 snRNAs. However, U2 snRNA with all uracils substituted with FUra (FU2) did not restore any splicing activity. Nondenaturing gel electrophoresis showed that FU2 failed to promote the assembly of spliceosome complexes. The ability of U2 snRNA to restore splicing in U2-depleted extracts increased as FUra content decreased but was still only 60% of control activity at 25% substitution of uracils with FUra. Addition of FU2 to nondepleted extracts caused strong inhibition of splicing accompanied by increased degradation of the pre-mRNA, suggesting that FU2 forms an inactive complex with a protein splicing factor that normally binds to the pre-mRNA. FU6 restored full splicing activity to U6-depleted extracts, but at a 5-fold higher concentration than U6 snRNA. These results demonstrate that the incorporation of FUra can impair the functions of catalytic RNA molecules.

L9 ANSWER 3 OF 21 MEDLINE on STN

ACCESSION NUMBER: 94377981 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8091213
TITLE: Complementation by SR proteins of pre-mRNA splicing reactions depleted of U1 snRNP.
AUTHOR: Crispino J D; Blencowe B J; Sharp P A
CORPORATE SOURCE: Center for Cancer Research, Massachusetts Institute of Technology, Cambridge 02139.
CONTRACT NUMBER: P30 CA14051 (NCI)
RO1-AI32486 (NIAID)
RO1-GM34277 (NIGMS)
SOURCE: Science, (1994 Sep 23) Vol. 265, No. 5180, pp. 1866-9.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941031
Last Updated on STN: 20000303
Entered Medline: 19941019

AB Individual small nuclear ribonucleoproteins (snRNPs) U1, U2, and U4/U6 were removed from nuclear extracts of HeLa cells by antisense affinity depletion. Addition of a highly purified preparation of SR proteins fully restored splicing activity in reactions depleted of U1 snRNP but did not reconstitute splicing in reactions depleted of the other snRNPs. Affinity selection experiments revealed that spliceosomes lacking U1 snRNA formed in the U1 snRNP-depleted reactions reconstituted with SR proteins. Thus, high concentrations of SR proteins facilitate the assembly of precursor messenger RNA (pre-mRNA) into a spliceosome in the absence of interactions with U1 snRNP.

L9 ANSWER 4 OF 21 MEDLINE on STN

ACCESSION NUMBER: 93268264 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7684489
TITLE: Uncoupling two functions of the U1 small nuclear ribonucleoprotein particle during in vitro splicing.
AUTHOR: Seiwert S D; Steitz J A
CORPORATE SOURCE: Department of Molecular Biophysics and Biochemistry, Howard Hughes Medical Institute, Yale University, Boyer Center for Molecular Medicine, New Haven, Connecticut 06536-0812.

CONTRACT NUMBER: GM26154 (NIGMS)
SOURCE: Molecular and cellular biology, (1993 Jun) Vol.
13, No. 6, pp. 3135-45.
Journal code: 8109087. ISSN: 0270-7306.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199306
ENTRY DATE: Entered STN: 19930702
Last Updated on STN: 20000303
Entered Medline: 19930623

AB To probe functions of the U1 small nuclear ribonucleoprotein particle (snRNP) during in vitro splicing, we have used unusual splicing substrates which replace the 5' splice site region of an adenovirus substrate with spliced leader (SL) RNA sequences from Leptomonas collosoma or *Caenorhabditis elegans*. In agreement with previous results (J.P. Bruzik and J.A. Steitz, Cell 62:889-899, 1990), we find that oligonucleotide-targeted RNase H destruction of the 5' end of U1 snRNA inhibits the splicing of a standard adenovirus splicing substrate but not of the SL RNA-containing substrates. However, use of an antisense 2'-O-methyl oligoribonucleotide that disrupts the first stem of U1 snRNA as well as stably sequestering positions of U1 snRNA involved in 5' and 3' splice site recognition inhibits the splicing of both the SL constructs and the standard adenovirus substrate. The 2'-O-methyl oligoribonucleotide is no more effective than RNase H pretreatment in preventing pairing of U1 with the 5' splice site, as assessed by inhibition of psoralen cross-link formation between the SL RNA-containing substrate and U1. The 2'-O-methyl oligoribonucleotide does not alter the protein composition of the U1 monoparticle or deplete the system of essential splicing factors. Native gel analysis indicates that the 2'-O-methyl oligoribonucleotide inhibits splicing by diminishing the formation of splicing complexes. One interpretation of these results is that removal of the 5' end of U1 inhibits base pairing in a different way than sequestering the same sequence with a complementary oligoribonucleotide. Alternatively, our data may indicate that two elements near the 5' end of U1 RNA normally act during spliceosome assembly; the extreme 5' end base pairs with the 5' splice site, while the sequence or structural integrity of stem I is essential for some additional function. It follows that different introns may differ in their use of the repertoire of U1 snRNP functions.

L9 ANSWER 5 OF 21 MEDLINE on STN
ACCESSION NUMBER: 93260006 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8491767
TITLE: Nucleoplasmic organization of small nuclear
ribonucleoproteins in cultured human cells.
AUTHOR: Matera A G; Ward D C
CORPORATE SOURCE: Department of Genetics, Yale University School of Medicine,
New Haven, Connecticut 06510.
CONTRACT NUMBER: GM-40115 (NIGMS)
HG-00246 (NHGRI)
SOURCE: The Journal of cell biology, (1993 May) Vol. 121,
No. 4, pp. 715-27.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199306
ENTRY DATE: Entered STN: 19930625
Last Updated on STN: 19980206
Entered Medline: 19930611

AB The organization of eight small nuclear ribonucleoproteins (the U1

, U2, U4, U5, and U6 RNAs previously studied by others and three additional snRNAs, U11, U12, and 7SK) has been investigated in cultured human cells by fluorescence in situ hybridization with antisense DNA and 2'-O-Me RNA oligonucleotides. Using highly sensitive digital imaging microscopy we demonstrate that all of these snRNAs are widespread throughout the nucleoplasm, but they are excluded from the nucleoli. In addition, the U2, U4, U5, U6, and U12 snRNAs are concentrated in discrete nuclear foci, known as coiled bodies, but U1 and 7SK are not. In addition to coiled bodies, a classic speckled pattern was observed in the nucleoplasm of monolayer-grown HeLa cells, whereas suspension-grown HeLa cells revealed a more diffuse nucleoplasmic labeling. Immunofluorescence staining using various snRNP-specific antisera shows complete agreement with that of their antisense snRNA oligonucleotide counterparts. Although U2 RNA is concentrated in coiled bodies, quantitation of the fluorescence signals from the U2 antisense probe reveals that the bulk of the U2 snRNP is located in the nucleoplasm. Furthermore, simultaneous visualization of the U2 snRNAs and the tandemly repeated U2 genes demonstrates that coiled bodies are not the sites of U2 transcription.

L9 ANSWER 6 OF 21 MEDLINE on STN
ACCESSION NUMBER: 93077666 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1447287
TITLE: Assembly and localization of the U1-specific snRNP C protein in the amphibian oocyte.
AUTHOR: Jantsch M F; Gall J G
CORPORATE SOURCE: Department of Embryology, Carnegie Institution, Baltimore, Maryland 21210.
CONTRACT NUMBER: GM 33397 (NIGMS)
SOURCE: The Journal of cell biology, (1992 Dec) Vol. 119, No. 5, pp. 1037-46.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M31166; GENBANK-M74319; GENBANK-M74320; GENBANK-M74321; GENBANK-M74322; GENBANK-M74323; GENBANK-M74324; GENBANK-X63892; GENBANK-X67016; GENBANK-Z23261
ENTRY MONTH: 199212
ENTRY DATE: Entered STN: 19930129
Last Updated on STN: 20000303
Entered Medline: 19921229

AB To study the intranuclear localization of the U1-specific snRNP C protein and its assembly into U1 snRNPs, we injected transcripts encoding a myc-tagged C protein into amphibian oocytes. The distribution of protein translated from the injected RNA was essentially the same in continuous and pulse-label experiments. In both cases the C protein localized within the germinal vesicle in those structures known to contain U1 snRNPs, namely the lampbrush chromosome loops and hundreds of extrachromosomal granules called snurposomes. Oocytes were also injected with an antisense oligodeoxynucleotide that caused truncation of U1 snRNA at the 5' end. In these oocytes, myc-tagged C protein localized normally in the germinal vesicle and could be immunoprecipitated together with truncated U1 snRNA. These experiments suggest that the C protein can enter the germinal vesicle on its own and there associate with previously assembled U1 snRNPs. In transfected tissue culture cells, the myc-tagged C protein localized within the nucleus in a speckled pattern similar to that of endogenous U1 snRNPs.

L9 ANSWER 7 OF 21 MEDLINE on STN
ACCESSION NUMBER: 93027140 MEDLINE

DOCUMENT NUMBER: PubMed ID: 1408748
TITLE: Antisense probes targeted to an internal domain in U2 snRNP specifically inhibit the second step of pre-mRNA splicing.
AUTHOR: Barabino S M; Sproat B S; Lamond A I
CORPORATE SOURCE: European Molecular Biology Laboratory, Heidelberg, Germany.
SOURCE: Nucleic acids research, (1992 Sep 11) Vol. 20, No. 17, pp. 4457-64.
Journal code: 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199210
ENTRY DATE: Entered STN: 19930122
Last Updated on STN: 19970203
Entered Medline: 19921030

AB Functional domains within the mammalian U2 snRNP particle that are required for pre-mRNA splicing have been analysed using antisense oligonucleotides. A comparison of the melting temperatures of duplexes formed between RNA and different types of antisense oligonucleotides has demonstrated that the most stable hybrids are formed with probes made of 2'-O-allyl RNA incorporating the modified base 2-aminoadenine. We have therefore used these 2'-O-allyl probes to target sequences within the central domain of U2 snRNA. Overlapping biotinylated 2'-O-allyl oligoribonucleotides complementary to the stem loop IIa region of U2 snRNA (nucleotides 54-72) specifically affinity selected U2 snRNA from HeLa nuclear extracts. These probes inhibited mRNA production in an in vitro splicing assay and caused a concomitant accumulation of splicing intermediates. Little or no inhibition of spliceosome assembly and 5' splice site cleavage was observed for all pre-mRNAs tested, indicating that the oligonucleotides were specifically inhibiting exon ligation. This effect was most striking with a 2'-O-allyl oligoribonucleotide complementary to U2 snRNA nucleotides 57-68. These results provide evidence for a functional requirement for U2 snRNP in the splicing mechanism occurring after spliceosome assembly.

L9 ANSWER 8 OF 21 MEDLINE on STN
ACCESSION NUMBER: 92329993 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1627829
TITLE: Transcription on lampbrush chromosome loops in the absence of U2 snRNA.
AUTHOR: Tsvetkov A; Jantsch M; Wu Z; Murphy C; Gall J G
CORPORATE SOURCE: Department of Embryology, Carnegie Institution, Baltimore, Maryland 21210.
CONTRACT NUMBER: GM 33397 (NIGMS)
SOURCE: Molecular biology of the cell, (1992 Mar) Vol. 3, No. 3, pp. 249-61.
Journal code: 9201390. ISSN: 1059-1524.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199208
ENTRY DATE: Entered STN: 19920904
Last Updated on STN: 19970203
Entered Medline: 19920820

AB The five small nuclear RNAs (snRNAs) involved in splicing occur on the loops of amphibian lampbrush chromosomes and in hundreds to thousands of extrachromosomal granules called B snurposomes. To assess the role of these snRNAs during transcription and to explore possible relationships between the loops and B snurposomes, we injected single-stranded antisense oligodeoxynucleotides (oligos) against U1 and U2 snRNA into toad and newt oocytes.

As shown before, antisense U1 and U2 oligos caused truncation of U1 and complete destruction of U2 snRNAs, respectively. However, injection of any oligo, regardless of sequence, brought on dramatic cytological changes, including shortening of the chromosomes and retraction of the lateral loops, with concomitant shutdown of polymerase II transcription, as well as disappearance of some or all of the B snurposomes. When injected oocytes were incubated for 12 h or longer in physiological saline, these changes were reversible; that is, the chromosomes lengthened, transcription (detected by 3H-UTP incorporation) resumed on newly extended lateral loops, and B snurposomes reappeared. In situ hybridization showed that loops and B snurposomes had negligible amounts of U2 snRNA after recovery from injection of the anti-U2 oligo, whereas these structures had normal levels of U2 snRNA after recovery from a control oligo. Thus, the morphological integrity of B snurposomes and lampbrush chromosome loops is not dependent on the presence of U2 snRNA. Because transcription occurs in the absence of U2 snRNA, we conclude that splicing is not required for transcription on lampbrush chromosome loops.

L9 ANSWER 9 OF 21 MEDLINE on STN
ACCESSION NUMBER: 91260692 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1646389
TITLE: Structural analyses of the 7SK ribonucleoprotein (RNP), the most abundant human small RNP of unknown function.
AUTHOR: Wassarman D A; Steitz J A
CORPORATE SOURCE: Department of Molecular Biophysics and Biochemistry, Howard Hughes Medical Institute, Yale University School of Medicine, New Haven, Connecticut 06510-8024.
CONTRACT NUMBER: GM26154 (NIGMS)
SOURCE: Molecular and cellular biology, (1991 Jul) Vol. 11, No. 7, pp. 3432-45.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199107
ENTRY DATE: Entered STN: 19910802
Last Updated on STN: 19970203
Entered Medline: 19910717

AB The human 7SK ribonucleoprotein (RNP) has been analyzed to determine its RNA secondary structure and protein constituents. HeLa cell 7SK RNA alone and within its RNP have been probed by chemical modification and enzymatic cleavage, and sites of modification or cleavage have been mapped by primer extension. The resulting secondary structure suggests that structural determinants necessary for capping (a 5' stem followed by the sequence AUPuUPuC) and nuclear migration (the sequence AUPuUPuC) of 7SK RNA may be similar to those for U6 small nuclear RNA (snRNA). It also supports existence of a 3' stem structure which could serve to self-prime cDNA synthesis during pseudogene formation. Oligonucleotide-directed RNase H digestion indicated regions of 7SK RNA capable of base pairing with other nucleic acids. Antisense 2'-O-methyl RNA oligonucleotides were used to affinity select the 7SK RNP from an in vivo 35S-labeled cell sonic extract and identify eight associated proteins of 83, 48, 45, 43, 42, 21, 18, and 13 kDa. 7SK RNA has extensive sequence complementarity to U4 snRNA, within the U4/U6 base pairing domain, and also to U11 snRNA. The possibility that the 7SK RNP is an unrecognized component of the pre-mRNA processing machinery is discussed.

L9 ANSWER 10 OF 21 MEDLINE on STN
ACCESSION NUMBER: 91114704 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1824936
TITLE: Mammalian nuclei contain foci which are highly enriched in components of the pre-mRNA splicing machinery.

AUTHOR: Carmo-Fonseca M; Tollervey D; Pepperkok R; Barabino S M;
Merdes A; Brunner C; Zamore P D; Green M R; Hurt E; Lamond
A I

CORPORATE SOURCE: European Molecular Biology Laboratory, Heidelberg, FRG.

SOURCE: The EMBO journal, (1991 Jan) Vol. 10, No. 1, pp.
195-206.

JOURNAL CODE: 8208664. ISSN: 0261-4189.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199103

ENTRY DATE: Entered STN: 19910329
Last Updated on STN: 19970203
Entered Medline: 19910301

AB The organization of the major snRNP particles in mammalian cell nuclei has been analysed by *in situ* labelling using snRNA-specific antisense probes made of 2'-OMe RNA. U3 snRNA is exclusively detected in the nucleolus while all the spliceosomal snRNAs are found in the nucleoplasm outside of nucleoli. Surprisingly, U2, U4, U5 and U6 snRNAs are predominantly observed in discrete nucleoplasmic foci. U1 snRNA is also present in foci but in addition is detected widely distributed throughout the nucleoplasm. An anti-peptide antibody specific for the non-snRNP splicing factor U2AF reveals it to have a similar distribution to U1 snRNA. Co-localization studies using confocal fluorescence microscopy prove that U2AF is present in the snRNA-containing foci. Antibody staining also shows the foci to contain snRNP-specific proteins and m3G-cap structures. The presence of major components of the nuclear splicing apparatus in foci suggests that these structures may play a role in pre-mRNA processing.

L9 ANSWER 11 OF 21 MEDLINE on STN

ACCESSION NUMBER: 91093166 MEDLINE

DOCUMENT NUMBER: PubMed ID: 1845976

TITLE: Biotinylated antisense methylphosphonate oligodeoxynucleotides. Inhibition of spliceosome assembly and affinity selection of U1 and U2 small nuclear RNPs.

AUTHOR: Temsamani J; Agrawal S; Pederson T

CORPORATE SOURCE: Cell Biology Group, The Worcester Foundation for Experimental Biology, Shrewsbury, Massachusetts 01545.

CONTRACT NUMBER: GM-21595-16 (NIGMS)

SOURCE: The Journal of biological chemistry, (1991 Jan 5)
Vol. 266, No. 1, pp. 468-72.
Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199102

ENTRY DATE: Entered STN: 19910322
Last Updated on STN: 19970203
Entered Medline: 19910214

AB Methylphosphonate (PC) backbone oligodeoxynucleotides complementary to the 5'-terminal nucleotides of U1 and U2 small nuclear (sn) RNAs do not elicit RNase H action under conditions in which natural (phosphodiester) oligodeoxynucleotides yield extensive RNase H cleavage. We show here that antisense PC oligonucleotides can mask sites in U1 and U2 snRNPs that are required for spliceosome formation. We further report that biotinylated derivatives of antisense PC oligos can be used for affinity selection of U1 and U2 snRNPs.

L9 ANSWER 12 OF 21 MEDLINE on STN

ACCESSION NUMBER: 91081326 MEDLINE

DOCUMENT NUMBER: PubMed ID: 2147994
TITLE: Sequence-specific affinity selection of mammalian splicing complexes.
AUTHOR: Ryder U; Sproat B S; Lamond A I
CORPORATE SOURCE: European Molecular Biology Laboratory, Heidelberg, FRG.
SOURCE: Nucleic acids research, (1990 Dec 25) Vol. 18, No. 24, pp. 7373-9.
Journal code: 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199101
ENTRY DATE: Entered STN: 19910322
Last Updated on STN: 19970203
Entered Medline: 19910129

AB Antisense oligonucleotides made of 2'-OMe RNA are shown to bind specifically and efficiently to targeted sites on pre-mRNA substrates, allowing affinity selection of splicing complexes using streptavidin/biotin chromatography. The position of probe binding to the pre-mRNA influences which type of splicing complex can be selected. The accessibility of pre-mRNA sequences to antisense probes changes during the course of the splicing reaction. U1, U2, U4, U5 and U6 snRNAs are all detected in affinity-selected mammalian splicing complexes. However, antisense oligonucleotides targeted to snRNAs can block the binding of specific snRNPs to pre-mRNA. Quantitative affinity selection analyses show that only a small fraction of snRNPs in a HeLa nuclear splicing extract participate in spliceosome formation.

L9 ANSWER 13 OF 21 MEDLINE on STN
ACCESSION NUMBER: 91045059 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2172924
TITLE: A 62,000 molecular weight spliceosome protein crosslinks to the intron polypyrimidine tract.
AUTHOR: Wang J; Pederson T
CORPORATE SOURCE: Cell Biology Group, Worcester Foundation for Experimental Biology, Shrewsbury, MA 01545, USA.
CONTRACT NUMBER: GM-21595-16 (NIGMS)
P30-12708-19
SOURCE: Nucleic acids research, (1990 Oct 25) Vol. 18, No. 20, pp. 5995-6001.
Journal code: 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199012
ENTRY DATE: Entered STN: 19910208
Last Updated on STN: 19970203
Entered Medline: 19901205

AB Incubation in HeLa nuclear extract of a 32P-labeled 61 nucleotide-long RNA corresponding to the lariat branch site/polypyrimidine tract/3' splice site of the first intron of human beta-globin pre-mRNA led to the crosslinking of a single protein of approximately 62,000 mol. weight (p62). p62 corresponds to a polypyrimidine tract-binding protein recently described by Garcia-Blanco et al. (Genes & Dev. 3: 1874-1886, 1989). Crosslinking of p62 to the 61 nt RNA was highly sequence specific. No p62 crosslinking was observed with a 60 nt pGEM vector RNA, a 63 nt RNA antisense to the 61-mer or a 72 nt U2 RNA sequence. p62 crosslinking to the 61 nt RNA was competed by unlabeled 61 nt RNA, by beta-globin pre-mRNA containing intron 1, and by poly(U) and poly(C), but was competed to a lesser extent or not at all by pGEM RNA, a beta-globin RNA lacking intron 1, or poly(A). Experiments with mutated RNAs revealed that neither the lariat branch site adenosine nor the 3' splice site were

required for p62 crosslinking to polypyrimidine tract-containing RNA. Elimination of the polypyrimidine tract reduced p62 crosslinking, as did mutation of a polypyrimidine tract UU dinucleotide to GA. However, replacement of a pyrimidine-rich tract immediately adjacent (3') to the lariat branch site with a 57% A + G pGEM vector RNA sequence also significantly reduced p62 crosslinking, indicating the involvement of both this pyrimidine-rich region and the classical polypyrimidine tract adjacent to the 3' splice site. The sites of protein interaction were further defined by RNase H protection experiments, the results of which confirmed the patterns of p62 crosslinking to mutant RNAs. p62 crosslinking was efficiently competed by a DNA oligonucleotide having the same sequence as the 61 nt RNA, showing that p62 requires neither ribose 2' OH groups nor uracil bases for its interaction with the polypyrimidine tract. p62 was not crosslinked to double-stranded 61 nt RNA. Q-Sepharose chromatography of HeLa nuclear extract yielded an unbound fraction (QU) in which p62 was the only polypyrimidine tract-crosslinkable protein and a bound fraction (QB) in which, surprisingly, several non-p62 proteins were crosslinkable to the polypyrimidine tract RNA. Yet, when the two Q-Sepharose fractions were combined, p62 strongly out-competed the otherwise-crosslinkable QB proteins for polypyrimidine tract RNA crosslinking. This indicates that p62 may have the highest affinity and/or crosslinking efficiency for the intron polypyrimidine tract of any HeLa nuclear protein.

L9 ANSWER 14 OF 21 MEDLINE on STN
ACCESSION NUMBER: 91004237 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2170025
TITLE: Targeted snRNP depletion reveals an additional role for mammalian U1 snRNP in spliceosome assembly.
AUTHOR: Barabino S M; Blencowe B J; Ryder U; Sproat B S; Lamond A I
CORPORATE SOURCE: European Molecular Biology Laboratory, Heidelberg, Federal Republic of Germany.
SOURCE: Cell, (1990 Oct 19) Vol. 63, No. 2, pp. 293-302.
Journal code: 0413066. ISSN: 0092-8674.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199011
ENTRY DATE: Entered STN: 19910117
Last Updated on STN: 19970203
Entered Medline: 19901119

AB HeLa cell nuclear splicing extracts have been prepared that are specifically and efficiently depleted of U1, U2, or U4/U6 snRNPs by antisense affinity chromatography using biotinylated 2'-OMe RNA oligonucleotides. Removal of each snRNP particle prevents pre-mRNA splicing but arrests spliceosome formation at different stages of assembly. Mixing extracts depleted for different snRNP particles restores formation of functional splicing complexes. Specific binding of factors to the 3' splice site region is still detected in snRNP-depleted extracts. Depletion of U1 snRNP impairs stable binding of U2 snRNP to the pre-mRNA branch site. This role of U1 snRNP in promoting stable preslicing complex formation is independent of the U1 snRNA-5' splice site interaction.

L9 ANSWER 15 OF 21 MEDLINE on STN
ACCESSION NUMBER: 89324084 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2526684
TITLE: Probing the structure and function of U2 snRNP with antisense oligonucleotides made of 2'-OMe RNA.
AUTHOR: Lamond A I; Sproat B; Ryder U; Hamm J
CORPORATE SOURCE: European Molecular Biology Laboratory, Heidelberg, Federal Republic of Germany.

SOURCE: Cell, (1989 Jul 28) Vol. 58, No. 2, pp. 383-90.
Journal code: 0413066. ISSN: 0092-8674.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M27897
ENTRY MONTH: 198909
ENTRY DATE: Entered STN: 19900309
Last Updated on STN: 19970203
Entered Medline: 19890907

AB We have used oligonucleotides made of 2'-OMe RNA to analyze the role of separate domains of U2 snRNA in the splicing process. We show that antisense 2'-OMe RNA oligonucleotides bind efficiently and specifically to U2 snRNP and demonstrate that masking of two separate regions of U2 snRNA can inhibit splicing by affecting different steps in the spliceosome assembly pathway. Masking the 5' terminus of U2 snRNA does not prevent U2 snRNP binding to pre-mRNA but blocks subsequent assembly of a functional spliceosome. By contrast, masking of U2 sequences complementary to the pre-mRNA branch site completely inhibits binding of pre-mRNA. Hybrid formation at the branch site complementary region also triggers a specific change which affects the 5' terminus of U2 snRNA.

L9 ANSWER 16 OF 21 MEDLINE on STN
ACCESSION NUMBER: 88217888 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2453054
TITLE: A sequence-specific conformational epitope on U1 RNA is recognized by a unique autoantibody.
AUTHOR: Deutscher S L; Keene J D
CORPORATE SOURCE: Department of Microbiology and Immunology, Duke University Medical Center, Durham, NC 27710.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1988 May) Vol. 85, No. 10, pp. 3299-303.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198806
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19880622

AB An autoantibody from a patient with lupus-overlap syndrome was found to bind a specific region of U1 RNA. By using RNA sequence analysis, immunoprecipitation, and competition experiments with in vitro synthesized fragments of U1 RNA, a region of 40 nucleotides representing a stem-loop secondary structure was found to be an immunoreactive domain. This antibody recognized a conformational epitope because neither the RNA stem nor the RNA loop alone was immunoprecipitable. Antisense U1 RNA, U1 DNA, and other small RNAs were not reactive with the antibody. While the origins of nucleic acid-binding antibodies are unknown, this RNA-specific autoantibody probably originated by direct presentation to the immune system or as an anti-idiotype against a more common U1 small nuclear ribonucleoprotein-specific autoantibody. Thus, these findings have implications for the mechanisms of autoimmune recognition and provide an immunological approach to probing RNA structure and protein-RNA interactions.

L9 ANSWER 17 OF 21 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 126:302364 CA
TITLE: Antisense, triplex-forming, or ribozyme oligonucleotides intracellular delivery using

oligonucleotide generator vector, triplex-forming
oligonucleotide screening, and applications in gene
regulation

INVENTOR(S): Noonberg, Sarah B.; Hunt, C. Anthony
PATENT ASSIGNEE(S): University of California, USA
SOURCE: U.S., 63 pp., Cont.-in-part of U.S. Ser. No. 138,666,
abandoned.
CODEN: USXXAM

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------------|------|----------|-----------------|--------------|
| US 5624803 | A | 19970429 | US 1994-324001 | 19941013 |
| CA 2173361 | AA | 19950420 | CA 1994-2173361 | 19941014 <-- |

PRIORITY APPLN. INFO.: US 1993-138666 B2 19931014
 AB The present invention encompasses improved methods and materials for the delivering of antisense, triplex, and/or ribozyme oligonucleotides intracellularly, and RNA polymerase III-based constructs termed "oligonucleotide generators" to accomplish the delivery of oligonucleotides. Also encompassed by the present invention are methods for screening oligonucleotide sequences that are candidates for triplex formation.

L9 ANSWER 18 OF 21 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 122:206988 CA

TITLE: Use of snRNA or mRNA promoter elements and 3' termination signals of plants for expression of genes in plants

INVENTOR(S): Filipowicz, Witold; Connelly, Sheila

PATENT ASSIGNEE(S): Ciba-Geigy A.-G., Switz.

SOURCE: PCT Int. Appl., 49 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|------|----------|-----------------|--------------|
| WO 9500652 | A1 | 19950105 | WO 1994-IB155 | 19940615 <-- |
| W: AU, BB, BG, BR, BY, CA, CN, CZ, FI, GE, HU, JP, KE, KG, KP, KR,
KZ, LK, LV, MD, MG, MN, MW, NO, NZ, PL, RO, RU, SD, SI, SK, TJ,
TT, UA, US, UZ, VN | | | | |
| RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE,
BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG | | | | |
| CA 2162925 | AA | 19950105 | CA 1994-2162925 | 19940615 <-- |
| AU 9468056 | A1 | 19950117 | AU 1994-68056 | 19940615 <-- |
| EP 703987 | A1 | 19960403 | EP 1994-916369 | 19940615 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE | | | | |
| US 6018103 | A | 20000125 | US 1995-564109 | 19951215 |
| PRIORITY APPLN. INFO.: | | | CH 1993-1828 | A 19930618 |
| | | | WO 1994-IB155 | W 19940615 |

AB The present invention relates to chimeric gene constructions comprising **snRNA** or mRNA promoter elements and 3' termination signals which are independent of the promoter element in functional combination with a structural gene which codes for a polypeptide, which is expressed, in a natural manner, from an mRNA gene, for sense RNA, for **anti-sense RNA** or for **snRNA**. In addition to this, the present invention relates to recombinant DNA mols. which encompass such chimeric gene constructs, and, in addition, to microorganisms which harbor these plasmids and to processes for preparing the said chimeric gene constructs and

plasmids. Finally, the invention relates to plant cells, to parts of plants which can be regenerated to form entire and preferably fertile plants, and to the plants themselves including their descendants, insofar as they express the chimeric genes according to the invention. The 3' termination signal was shown to result in formation of a correct snRNA 3' end irresp. of whether the transcription was initiated by an snRNA promoter element or an mRNA promoter element. Transcripts whose synthesis was initiated by a plant snRNA promoter element could be correctly polyadenylated (and spliced, no data). Initiation of transcription on an snRNA promoter element did not require the presence of an snRNA-encoding region and/or of a 3' box.

L9 ANSWER 19 OF 21 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 117:63902 CA
TITLE: Chimeric antisense RNAs
AUTHOR(S): Izant, Jonathan G.
CORPORATE SOURCE: Dep. Genet., Yale Sch. Med., New Haven, CT, 16510, USA
SOURCE: Raven Press Series on Molecular and Cellular Biology (1992), 1(Gene Regul.), 183-95
CODEN: RPSBEH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Chimeric antisense RNAs were constructed by combining structural RNA genes with antisense DNA fragments in fusion genes. **Antisense** RNA vectors based on U2 RNA and tRNA genes produced chimeric RNA with the stability of the structural RNA and **antisense** activity.

L9 ANSWER 20 OF 21 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 113:110014 CA
TITLE: Specific regulation of gene expression by antisense, sense and antigenic nucleic acids
AUTHOR(S): Helene, Claude; Toulme, Jean Jacques
CORPORATE SOURCE: Lab. Biophys., Mus. Natl. Histoire Nat., Paris, 75231/05, Fr.
SOURCE: Biochimica et Biophysica Acta, Gene Structure and Expression (1990), 1049(2), 99-125
CODEN: BBGSD5; ISSN: 0167-4781
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 241 refs. DNA as a target for oligonucleotides, **antisense** nucleic acid complementary to U **snRNA** and pre-mRNA and their effect on splicing, mRNA as a target of **antisense** nucleic acids and their effect on translation and transcript steady state levels, proteins as targets, and irreversible reactions induced by derivatized oligonucleotides and ribozyme are discussed.

L9 ANSWER 21 OF 21 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:60105 SCISEARCH
THE GENUINE ARTICLE: TP243
TITLE: Efficient hammerhead ribozyme-mediated cleavage of the structured hepatitis B virus encapsidation signal in vitro and in cell extracts, but not in intact cells
AUTHOR: Beck J (Reprint); Nassal M
CORPORATE SOURCE: UNIV HEIDELBERG, ZENTRUM MOLEK BIOL, D-69120 HEIDELBERG, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: NUCLEIC ACIDS RESEARCH, (25 DEC 1995) Vol. 23, No. 24, pp. 4954-4962.
ISSN: 0305-1048.
PUBLISHER: OXFORD UNIV PRESS UNITED KINGDOM, WALTON ST JOURNALS DEPT, OXFORD, ENGLAND OX2 6DP.
DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 50
ENTRY DATE: Entered STN: 1996
Last Updated on STN: 1996
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Hepatitis B virus (HBV), the causative agent of B-type hepatitis in man, is a small enveloped DNA virus that replicates through reverse transcription of an RNA intermediate, the terminally redundant RNA pregenome. An essential highly conserved cis-element present twice on this RNA is the encapsidation signal epsilon, a stem-loop structure that is critical for pregenome packaging and reverse transcription, epsilon is hence an attractive target for antiviral therapy. Its structure, however, is a potential obstacle to antivirals whose action depends on hybridization, e.g. ribozymes. Here we demonstrate effective in vitro cleavage inside epsilon by hammerhead ribozymes containing flanking sequences complementary to an adjacent less structured region. Upon co-transfection with a HBV expression construct corresponding ribozymes embedded in a U6 snRNA context led to a significant, though modest, reduction in the steady-state level of HBV pregenomes. Inactive ribozyme mutants revealed that antisense effects contributed substantially to this reduction, however, efficient epsilon cleavage by the intracellularly expressed ribozymes was observed in Mg²⁺-supplemented cell lysates. Artificial HBV pregenomes carrying the ribozymes in cis and model RNAs lacking all HBV sequences except epsilon exhibited essentially the same behaviour. Hence, neither the absence of co-localization of ribozyme and target nor a viral component, but rather a cellular factor(s), is responsible for the strikingly different ribozyme activities inside cells and in cellular extracts.

=> s construct? or vector? or cassette?
L10 2015441 CONSTRUCT? OR VECTOR? OR CASSETTE?

=> s construct? or vector? or cassette? or plasmid?
L11 2316686 CONSTRUCT? OR VECTOR? OR CASSETTE? OR PLASMID?

=> d his

(FILE 'HOME' ENTERED AT 19:25:13 ON 10 APR 2006)

FILE 'MEDLINE, BIOSIS, CA, EMBASE, SCISEARCH' ENTERED AT 19:25:22 ON 10 APR 2006

L1 25343 F (U1 OR U2 OR SNRRNA)
L2 25343 S (U1 OR U2 OR SNRRNA)

FILE 'MEDLINE, BIOSIS, CA, EMBASE, SCISEARCH' ENTERED AT 19:25:59 ON 10 APR 2006

L3 25343 S (U1 OR U2 OR SNRRNA)
L4 141392 S ANTISENSE OR ANTI-SENSE
L5 107 S L3 (2N) L4
L6 443 S L3 (P) L4
L7 290 S L3 (S) L4
L8 97 DUP REM L7 (193 DUPLICATES REMOVED)
L9 21 S L8 AND (PD<=1995)
L10 2015441 S CONSTRUCT? OR VECTOR? OR CASSETTE?
L11 2316686 S CONSTRUCT? OR VECTOR? OR CASSETTE? OR PLASMID?

=> s l4 and l11
L12 33758 L4 AND L11

=> s l11 (p) 14 (p) (multipl? or ((more than (n) (1 or one)) or (at least (n) (two or 2)) or (two or 2)))
UNMATCHED LEFT PARENTHESIS 'P) (MULTIPL?)

The number of right parentheses in a query must be equal to the number of left parentheses.

=> s l11 (p) 14 (p) (multipl? or ((more than (n) (1 or one)) or (at least (n) (two or 2)) or (two or 2)))
3 FILES SEARCHED...

L13 10556 L11 (P) L4 (P) (MULTIPL? OR ((MORE THAN (N) (1 OR ONE)) OR (AT LEAST (N) (TWO OR 2)) OR (TWO OR 2)))

=> s l11 (p) 14 (p) (multipl? or ((more than (n) (1 or one)) or (at least (n) (two or 2))))
4 FILES SEARCHED...

L14 1292 L11 (P) L4 (P) (MULTIPL? OR ((MORE THAN (N) (1 OR ONE)) OR (AT LEAST (N) (TWO OR 2))))

=> s l11 (s) 14 (s) (multipl? or ((more than (n) (1 or one)) or (at least (n) (two or 2))))
4 FILES SEARCHED...

L15 130 L11 (S) L4 (S) (MULTIPL? OR ((MORE THAN (N) (1 OR ONE)) OR (AT LEAST (N) (TWO OR 2))))

=> s l15 (py<=1995)

MISSING OPERATOR 'L90 (PY<=1995'

The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s l15 and (py<=1995)

1 FILES SEARCHED...

L16 35 L15 AND (PY<=1995)

=> dup rem l16

PROCESSING COMPLETED FOR L16

L17 14 DUP REM L16 (21 DUPLICATES REMOVED)

=> d l17 ibib abs 1-14

L17 ANSWER 1 OF 14 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 135:41842 CA

TITLE: Vector with multiple target response elements
affecting gene expression for inhibition of viral
replication

INVENTOR(S): Lisziewicz, Julianna

PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA

SOURCE: U.S., 33 pp., Cont.-in-part of U.S. Ser. No. 69,476.

CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|--|------|----------|-----------------|--------------|
| US 6245560 | B1 | 20010612 | US 1994-245506 | 19940518 |
| US 467407 | A0 | 19900801 | US 1990-467407 | 19900118 <-- |
| US 596299 | A0 | 19910801 | US 1990-596299 | 19901015 <-- |
| CA 2190375 | AA | 19951123 | CA 1995-2190375 | 19950518 <-- |
| WO 9531477 | A1 | 19951123 | WO 1995-US5955 | 19950518 <-- |
| W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI,
GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD,
MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ,
TT, UA | | | | |
| RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT,
LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE,
SN, TD, TG | | | | |

| | | | | |
|---|----|----------|----------------|--------------|
| AU 9525874 | A1 | 19951205 | AU 1995-25874 | 19950518 <-- |
| AU 703362 | B2 | 19990325 | | |
| EP 759936 | A1 | 19970305 | EP 1995-920416 | 19950518 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE | | | | |
| PRIORITY APPLN. INFO.: | | | | |
| US 1990-467407 B2 19900118 | | | | |
| US 1990-596299 B1 19901015 | | | | |
| US 1993-69476 B2 19930601 | | | | |
| US 1994-245506 A 19940518 | | | | |
| WO 1995-US5955 W 19950518 | | | | |

AB The present invention relates to viral inhibition, particularly HIV inhibition, by DNA sequences including multiple target response elements. The DNA construct of the present invention comprises a vector and a promoter operably linked to at least one target response element and, optionally, to other viral inhibitory elements, so that the elements are transcribed in tandem. Vectors containing the HIV-1 LTR (from which the neg. regulatory element is deleted) linked to 1-50 copies of the HIV-1 tat activation response element (tar) cDNA were prepared COS-1 cells containing these plasmids as well as a plasmid containing the tat gene and one containing the chloramphenicol acetyltransferase gene, both controlled by the HIV-1 LTR were prepared Expression of the reporter gene was inversely proportional to the number of response elements present. The DNA construct can be used in the treatment of viral infections, HIV in particular, by obtaining cells from an HIV-infected patient, transforming the cells with the construct and administering the transformed cells to the patient. The protective gene product will only be expressed if the cell becomes infected and a viral protein is made.

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 2 OF 14 CA COPYRIGHT 2006 ACS on STN
 ACCESSION NUMBER: 124:106632 CA
 TITLE: Vectors with multiple HIV-1 TAR sequences inhibiting HIV-1 gene expression
 INVENTOR(S): Lisziewicz, Julianne
 PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
 SOURCE: PCT Int. Appl., 53 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 4
 PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|--|------|----------|-----------------|--------------|
| WO 9531477 | A1 | 19951123 | WO 1995-US5955 | 19950518 <-- |
| W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI,
GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD,
MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ,
TT, UA | | | | |
| RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT,
LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE,
SN, TD, TG | | | | |
| US 6245560 | B1 | 20010612 | US 1994-245506 | 19940518 |
| AU 9525874 | A1 | 19951205 | AU 1995-25874 | 19950518 <-- |
| AU 703362 | B2 | 19990325 | | |
| EP 759936 | A1 | 19970305 | EP 1995-920416 | 19950518 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE | | | | |
| PRIORITY APPLN. INFO.: | | | | |
| US 1994-245506 A 19940518 | | | | |
| US 1990-467407 B2 19900118 | | | | |
| US 1990-596299 B1 19901015 | | | | |
| US 1993-69476 B2 19930601 | | | | |
| WO 1995-US5955 W 19950518 | | | | |

AB Viral inhibition, particularly HIV inhibition, is effected by use of DNA

constructs which include multiple target response elements. A DNA construct for inhibition of HIV-1 gene expression comprises a vector and a primate lentivirus long terminal repeat (LTR) promoter operably linked to ≥2 target DNA response elements encoding HIV-1 tat activation response (TAR) elements and, optionally, to other viral inhibitory elements so that these elements are transcribed in tandem. The tat protein of HIV transactivates viral gene expression and is essential for expression of viral products; its transcriptional effect is evidently mediated through interaction with the TAR region of viral RNA. TAR RNA, produced in large amounts, might therefore serve as a competitive inhibitor of tat function. The DNA construct can be used in treatment of HIV infections by obtaining cells from an HIV-infected patient, transforming the cells with the construct, and administering the transformed cells to the patient. The protective gene product will only be expressed if the cell becomes infected and a viral protein is made. Thus, in COS cells cotransfected with HIV and a DNA construct containing 0-50 TAR sequences, the inhibition of transactivation (HIV-LTR-directed gene expression) paralleled the amount of TAR transcripts produced, as shown by Northern blotting expts. An antisense tat gene construct, antitat, inhibited HIV-1 replication when transduced into peripheral blood mononuclear cells from AIDS patients.

L17 ANSWER 3 OF 14 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER:

124:23311 CA

TITLE:

Inhibition of HIV-1 multiplication in mammalian cells with packaging signal and/or gag gene antisense RNA

INVENTOR(S):

Joshi-Sukhwal, Sadna

PATENT ASSIGNEE(S):

Can.

SOURCE:

PCT Int. Appl., 26 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|------|----------|-----------------|--------------|
| WO 9527783 | A1 | 19951019 | WO 1995-CA190 | 19950405 <-- |
| W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT | | | | |
| RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG | | | | |
| AU 9521336 | A1 | 19951030 | AU 1995-21336 | 19950405 <-- |
| PRIORITY APPLN. INFO.: | | | US 1994-223560 | A 19940406 |
| | | | WO 1995-CA190 | W 19950405 |

AB A method of inhibiting human immunodeficiency virus type 1 (HIV-1) in a mammal using mammalian cells, particularly, human CD4-containing lymphocytes, which express chimeric RNA mols. containing HIV-1 ψ signal and/or Gag coding sequences in antisense orientation. HIV-1 production was delayed up to 30 days when compared with control cells lacking the test DNA sequences. Retroviral vectors, e.g. those derived from Moloney murine leukemia virus, expressing the chimeric RNA mols. are provided.

L17 ANSWER 4 OF 14 MEDLINE on STN

DUPPLICATE 1

ACCESSION NUMBER: 96013753 MEDLINE

DOCUMENT NUMBER: PubMed ID: 7474070

TITLE:

A nonproducer, interfering human immunodeficiency virus (HIV) type 1 provirus can be transduced through a murine leukemia virus-based retroviral vector: recovery of an anti-HIV mouse/human pseudotype retrovirus.

AUTHOR: Federico M; Nappi F; Ferrari G; Chelucci C; Mavilio F;

CORPORATE SOURCE: Verani P
Laboratory of Virology, Istituto Superiore di Sanita, Rome,
Italy.
SOURCE: Journal of virology, (1995 Nov) Vol. 69, No. 11,
pp. 6618-26.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199512
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19970203
Entered Medline: 19951201

AB The expression of a human immunodeficiency virus (HIV) type 1 provirus (F12-HIV) cloned from a nonproducer, chronically infected CD4 down-regulated Hut-78 cell clone (F12) does not lead to the formation of viral particles and, upon transfection in HeLa CD4+ cells, confers resistance to HIV superinfection without affecting the CD4 receptor exposure. In an attempt to transfer the anti-HIV properties of F12-HIV into human primary cell, we constructed a Moloney murine leukemia virus-based retroviral vector containing an F12-HIV genome lacking the 3' long terminal repeat and part of the nef gene, which was expressed under the control of its 5' long terminal repeat. The F12-HIV genome was inserted in the orientation opposite to that of the murine leukemia virus transcriptional unit and was designated the N2/F12-HIV nef-antisense vector. Lymphoblastoid CEMss cells, as well as human peripheral blood lymphocytes, were successfully transduced by the recombinant retrovirus emerging from the producer PA317 clones. CEMss clones expressing the F12-HIV nef-antisense vector became resistant to HIV superinfection even at the highest utilized multiplicity of infection (10(5) 50% tissue culture infective doses per 10(6) cells). In transduced CEMss cells the viral interference induced by the F12-HIV expression is not due to CD4 HIV receptor down-regulation. Nonproducer, interfering HIV proviruses transduced into retroviral vectors may, therefore, provide an alternative strategy for the protection of CD4+ human primary cells from HIV infection, which strategy may be used in designating a safe and efficient gene therapy protocol for patients with AIDS.

L17 ANSWER 5 OF 14 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 96019255 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7593282
TITLE: Targeted disruption of the Dictyostelium myosin essential light chain gene produces cells defective in cytokinesis and morphogenesis.
AUTHOR: Chen T L; Kowalczyk P A; Ho G; Chisholm R L
CORPORATE SOURCE: Department of Cell and Molecular Biology, Northwestern University Medical School, Chicago, IL 60611, USA.
CONTRACT NUMBER: GM 39264 (NIGMS)
SOURCE: Journal of cell science, (1995 Oct) Vol. 108 (Pt 10), pp. 3207-18.
Journal code: 0052457. ISSN: 0021-9533.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199512
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19960124
Entered Medline: 19951218

AB We have previously demonstrated that the myosin essential light chain (ELC) is required for myosin function in a Dictyostelium cell line, 7-11, in which the expression of ELC was inhibited by antisense RNA

overexpression. We have now disrupted the gene encoding the ELC (*mlcE*) in *Dictyostelium* by gene targeting. The *mlcE*- mutants provide a clean genetic background for phenotypic analysis and biochemical characterization by removing complications arising from the residual ELC present in 7-11 cells, as well as the possibility of mutations due to insertion of the antisense construct at multiple sites in the genome. The *mlcE*- mutants, when grown in suspension, exhibited the typical multinucleate phenotype observed in both myosin heavy chain mutants and 7-11 cells. This phenotype was rescued by introducing a construct that expressed the wild-type *Dictyostelium* ELC cDNA. Myosin purified from the *mlcE*- cells exhibited significant calcium ATPase activity, but the actin-activated ATPase activity was greatly reduced. The results obtained from the *mlcE*- mutants strengthen our previous conclusion based on the antisense cell line 7-11 that ELC is critical for myosin function. The proper localization of myosin in *mlcE*- cells suggests that its phenotypic defects primarily arise from defective contractile function of myosin rather than its mislocalization. The enzymatic defect of myosin in *mlcE*- cells also suggests a possible mechanism for the observed chemotactic defect of *mlcE*- cells. We have shown that while *mlcE*- cells were able to respond to chemoattractant with proper directionality, their rate of movement was reduced. During chemotaxis, proper directionality toward chemoattractant may depend primarily on proper localization of myosin, while efficient motility requires contractile function. In addition, we have analyzed the morphogenetic events during the development of *mlcE*- cells using lacZ reporter constructs expressed from cell type specific promoters. By analyzing the morphogenetic patterns of the two major cell types arising during *Dictyostelium* development, prespore and prestalk cells, we have shown that the localization of prespore cells is more susceptible to the loss of ELC than prestalk cells, although localization of both cell types is abnormal when developed in chimeras formed by mixing equal numbers of wild-type and mutant cells. These results suggest that the morphogenetic events during *Dictyostelium* development have different requirements for myosin.

L17 ANSWER 6 OF 14 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1995:152530 SCISEARCH

THE GENUINE ARTICLE: QJ600

TITLE: LOSS OF THE METASTATIC PHENOTYPE IN MURINE CARCINOMA-CELLS EXPRESSING AN ANTISENSE RNA TO THE INSULIN-LIKE GROWTH-FACTOR RECEPTOR

AUTHOR: LONG L (Reprint); RUBIN R; BASERGA R; BRODT P

CORPORATE SOURCE: MCGILL UNIV, DEPT SURG, MONTREAL, PQ H3A 1A4, CANADA; MCGILL UNIV, DEPT ONCOL, MONTREAL, PQ H3A 1A4, CANADA; ROYAL VICTORIA HOSP, MONTREAL, PQ H3A 1A4, CANADA; THOMAS JEFFERSON UNIV, JEFFERSON MED COLL, DEPT PATHOL & CELL BIOL, PHILADELPHIA, PA 19107; THOMAS JEFFERSON UNIV, JEFFERSON CANC INST, PHILADELPHIA, PA 19107

COUNTRY OF AUTHOR: CANADA; USA

SOURCE: CANCER RESEARCH, (1 MAR 1995) Vol. 55, No. 5, pp. 1006-1009.

ISSN: 0008-5472.

PUBLISHER: AMER ASSOC CANCER RESEARCH, PUBLIC LEDGER BLDG, SUITE 816, 150 S. INDEPENDENCE MALL W., PHILADELPHIA, PA 19106.

DOCUMENT TYPE: Note; Journal

FILE SEGMENT: LIFE; CLIN

LANGUAGE: English

REFERENCE COUNT: 25

ENTRY DATE: Entered STN: 1995
Last Updated on STN: 1995

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The ability of malignant cells to form metastases in secondary sites remains a major obstacle to the curative treatment of cancer. Previously,

we identified type 1 insulin like growth factor (IGF-1) as a paracrine mitogen for highly metastatic murine carcinoma, H-59 cells. Here the role of IGF-I and its receptor (IGF-1R) in metastasis was further investigated using H-59 cells transfected with a plasmid vector expressing IGF-1R cDNA in the antisense orientation. The transfectants had a markedly reduced expression of IGF-1R and lost the ability to respond to IGF-1 in vitro. When injected in vivo, either directly into the microvasculature of the liver or lung (experimental metastasis) or s.c. to allow the growth of primary local tumors (spontaneous metastasis), these cells did not give rise to any metastases under conditions which allowed wild-type or control transfectants to form multiple hepatic and pulmonary metastases. The results demonstrate that the IGF-1R can play a critical role in the regulation of carcinoma metastasis.

L17 ANSWER 7 OF 14 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 95204963 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7897244
TITLE: A simple assay for examining the effect of transiently expressed genes on programmed cell death.
AUTHOR: Memon S A; Petrak D; Moreno M B; Zacharchuk C M
CORPORATE SOURCE: Laboratory of Immune Cell Biology, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892-1152.
SOURCE: Journal of immunological methods, (1995 Mar 13)
Vol. 180, No. 1, pp. 15-24.
Journal code: 1305440. ISSN: 0022-1759.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 19950504
Last Updated on STN: 19950504
Entered Medline: 19950425
AB Programmed cell death (PCD) has been observed in a wide variety of cell types in response to physiologic signals or types of stress. How these stimuli trigger PCD, and whether there is a common PCD signal transduction pathway, is not clear. As more genes are described that may participate in or regulate PCD, an assay system in which gene products can easily be introduced and/or modulated would be of great value. To avoid the generation and screening of multiple individual stable cell transfectants, a simple transient transfection death assay has been developed. 2B4.11, a murine T cell hybridoma, was transfected by electroporation with a constitutively active beta-galactosidase reporter gene and the cells were incubated in culture medium or with a PCD-inducing stimulus. The amount of beta-galactosidase activity remaining in the intact cells at the end of the culture period represented only viable transfected cells. Bcl-2 was chosen to examine whether this system would be useful to study the effect of transiently transfected genes since it blocks PCD in a number of experimental systems. Consistent with data obtained using stable transfectants, transient expression of Bcl-2 in 2B4.11 completely protected cells from glucocorticoid- and cytotoxic agent-induced PCD. This protection from death was confirmed at the individual cell level by the transient co-expression of a class I Ld surface antigen and flow cytometric analysis. Some of the advantages of the transient transfection death assay described here are; (1) the simple and sensitive beta-galactosidase assay, (2) the rapidity of the assay, (3) the ability to perform conventional viability assays to monitor treatment-induced cytotoxicity, (4) multiple gene products can be tested alone, and in combination, (5) antisense or dominant negative approaches can be used, and (6) the adaptability of this assay system to other cell types, transfection techniques, or reporter and expression vectors. The transient transfection death assay should make it

easier to identify and order important steps in the PCD signal transduction pathways.

L17 ANSWER 8 OF 14 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 96060830 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7590329
TITLE: Versatile, multi-featured plasmids for high-level expression of heterologous genes in *Escherichia coli*: overproduction of human and murine cytokines.
AUTHOR: Mertens N; Remaut E; Fiers W
CORPORATE SOURCE: Laboratory of Molecular Biology, University of Gent, Belgium.
SOURCE: Gene, (1995 Oct 16) Vol. 164, No. 1, pp. 9-15.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199512
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19980206
Entered Medline: 19951212
AB We describe the construction, expression characteristics and some applications of a versatile dual-promoter expression plasmid for heterologous gene expression in *Escherichia coli* which contains both lambda pL and PT7 promoters. Furthermore, the plasmid is optimized to allow the expression of mature coding sequences without compromising the strength of the highly efficient PT7 or of the T7g10 ribosome-binding site. The effect of the naturally occurring RNA loops at both the 5' and 3' ends of the T7g10 mRNA on expression was also examined. A double T7 RNA polymerase transcription terminator was inserted to ensure more reliable transcription termination and a higher expression level of the preceding gene. Further improvements involve a clockwise orientation of the promoters to minimize read-through transcription from **plasmid** promoters, a largely extended **multiple cloning site**, an **antisense** phage T3 promoter and a phage f1-derived, single-stranded replication origin. Variants of this plasmid allow for the production of fusion proteins with part of T7g10, a hexahistidine peptide and an enterokinase recognition site. The potential of these expression vectors is demonstrated by comparing the expression levels of a number of mammalian cytokines (human tumor necrosis factor, human immune interferon, human and murine interleukins 2, murine interleukin 4 and murine fibroblast interferon), using these expression plasmids.

L17 ANSWER 9 OF 14 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 120:24763 CA
TITLE: Highly identical cassettes of gene regulatory elements, genomically repetitive and present in RNA
AUTHOR(S): Nemer, Martin; Bai, Guang; Stuebing, Elizabeth W.
CORPORATE SOURCE: Inst. Cancer Res., Fox Chase Cancer Cent., Philadelphia, PA, 19111, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1993), 90(22), 10851-5
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A region in the first intron of a metallothionein-encoding gene of the sea urchin *Strongylocentrotus purpuratus* (SpMTA gene) regulates its 5' promoter activity. Within this region is a 290-bp cassette of six sequence motifs that are present in other genes in this species and posited to operate as regulatory elements. The cassette, present at high multiplicity in the genome, was used to screen genomic DNA clones. Of these, six diverse individuals were partially sequenced and found to have

segments 94% identical to the 290-bp cassette in the spMTA gene. Their next 80 bp diverged from the SpMTA sequence but were highly identical among the six non-SpMTA clones and contained an addnl. regulatory motif. These diverse clones thus contained 370-bp cassettes of an overall 94% sequence identity and an apparent content of seven regulatory elements. The regulatory cassettes were transposon-like, insofar as the termini of the highly identical regions consisted of 24- to 25-bp inverted repeats, bracketed by 6- to 9-bp direct repeats in the divergent regions. In addition to being in transcripts of the SpMTA intron, the cassette was found in other sea urchin embryo poly(A)+ RNAs, in eggs and embryos, and enriched in pluteus ectoderm. The cassette sequence was present in moderate abundance in transcripts in both sense and antisense orientation. The authors report the presence of a transposon-like cassette of regulatory elements that is also represented in RNA, which potentially could function differently from previously described transposons.

L17 ANSWER 10 OF 14 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 93207781 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8457354
TITLE: Improved design of riboprobes from pBluescript and related vectors for *in situ* hybridization.
AUTHOR: Witkiewicz H; Bolander M E; Edwards D R
CORPORATE SOURCE: Department of Orthopedics, Mayo Clinic, Rochester, MN 55905.
SOURCE: BioTechniques, (1993 Mar) Vol. 14, No. 3, pp. 458-63.
JOURNAL code: 8306785. ISSN: 0736-6205.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199304
ENTRY DATE: Entered STN: 19930514
Last Updated on STN: 20000303
Entered Medline: 19930423

AB The pBluescript family of plasmids and phagemids are sophisticated multi-purpose cloning vectors that allow convenient production of single-stranded sense and anti-sense RNA probes corresponding to DNA sequences inserted into a large multiple cloning site array. We have observed that in many applications sense (control) probes generated from genes cloned into pBluescript II KS(-) give high background signals on *in situ* hybridization to human tissue sections. Our studies indicate that this spurious hybridization is due to sequences contained within both strands of the multiple cloning site between the SmaI and SacI sites that are similar to human 28S rRNA. This information is useful in construct design in order to minimize nonspecific background problems, as demonstrated by *in situ* hybridization of sense and anti-sense probes corresponding to a portion of human stromelysin-3 to sections of human lung carcinoma.

L17 ANSWER 11 OF 14 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 94003941 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7916586
TITLE: Simultaneous inhibition of two tomato fruit cell wall hydrolases, pectinmethylesterase and polygalacturonase, with antisense gene constructs.
AUTHOR: Pear J R; Sanders R A; Summerfelt K R; Martineau B; Hiatt W R
CORPORATE SOURCE: Calgene, Inc., Davis, California.
SOURCE: Antisense research and development, (1993 Summer) Vol. 3, No. 2, pp. 181-90.
JOURNAL code: 9110698. ISSN: 1050-5261.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
OTHER SOURCE: GENBANK-L05008; GENBANK-L05009; GENBANK-L05010;
GENBANK-L05011; GENBANK-L05012; GENBANK-L05013;
GENBANK-L05014; GENBANK-L08748; GENBANK-M97169;
GENBANK-S66607

ENTRY MONTH: 199311
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 19950206
Entered Medline: 19931105

AB The cloning and sequencing of two cDNAs representing pectinmethyl esterase (PME) RNAs from tomato fruit is reported. The clones were used to construct chimeric antisense PME genes designed for high-level constitutive expression in plants. A full-length antisense PME gene construct, in conjunction with a chimeric antisense polygalacturonase gene, was introduced into tomato plants via Agrobacterium-mediated transformation. Simultaneous and significant reduction in the mRNA and protein levels of these normally highly abundant cell wall hydrolases of the pectin degradation pathway were observed in ripe fruit of transformants. Thus, antisense gene constructs in plants can be used to block multiple steps in metabolic pathways simultaneously.

L17 ANSWER 12 OF 14 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1992:318846 BIOSIS
DOCUMENT NUMBER: PREV199243019571; BR43:19571
TITLE: INHIBITION OF HIV-1 RECEPTOR REPLICATION BY BLOCKING TAT EXPRESSION AND ACTIVITY WITH ANTISENSE RNA EXPRESSING PLASMIDS AND MULTIPLE TAR CONSTRUCT.
AUTHOR(S): CHANG H-K [Reprint author]; LISZIEWICZ J; FIORELLI V; GALLO R C; ENSOLI B
CORPORATE SOURCE: LAB TUMOR CELL BIOL, NATL CANCER INST, NATL INST HEALTH, BETHESDA, MD 20892, USA
SOURCE: Journal of Cellular Biochemistry Supplement, (1992) No. 16 PART E, pp. 76.
Meeting Info.: KEYSTONE SYMPOSIUM ON PREVENTION AND TREATMENT OF AIDS, KEYSTONE, COLORADO, USA, MARCH 27-APRIL 3, 1992. J CELL BIOCHEM SUPPL.
ISSN: 0733-1959.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 30 Jun 1992
Last Updated on STN: 30 Jun 1992

L17 ANSWER 13 OF 14 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 118:206202 CA
TITLE: Vectors with segmented multiple cloning sites (SMCS) for easy monitoring of restriction digests and post-cloning orientation reversal of genes
AUTHOR(S): Georges, Fawzy; Hussain, Atta; Papish, Bob
CORPORATE SOURCE: Plant Biotechnol. Inst., Natl. Res. Coun. Canada, Saskatoon, SK, S7N 0W9, Can.
SOURCE: Gene (1992), 111(1), 27-33
CODEN: GENED6; ISSN: 0378-1119
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A novel approach is described, through which the transcriptional orientation of cloned genes is manipulated without further subcloning. This is achieved through a restriction/ligation process (without changing the test tube) leading to approx. 1:1 mixture of both possible orientations. The plasmid containing the reversed orientation is easily distinguished from

the wild type by built-in restriction-indicator sites for SpeI. Addnl., a large marker fragment is incorporated into the segmented multiple cloning site (SMCS) regions to facilitate monitoring the progress and/or efficiency of vector restrictions. The marker fragment is released upon appropriate double digestion, thus acting as an indicator for complete restriction-enzyme digestion. The authors equipped some existing plasmids, commonly used in mol. biol., with these features and demonstrated their superiority to plasmids having regular MCS sequences.

L17 ANSWER 14 OF 14 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 113:185786 CA

TITLE: Inhibition of the activity of chloramphenicol acetyltransferase (CAT) in transgenic tobacco plants by the synthesis of antisense RNA

AUTHOR(S): Kanevskii, I. F.; Nagy, F.

CORPORATE SOURCE: Inst. Bot. im. Kholodnogo, Kiev, USSR

SOURCE: Doklady Akademii Nauk SSSR (1990), 312(4), 981-3, 1 plate [Genet.]

CODEN: DANKAS; ISSN: 0002-3264

DOCUMENT TYPE: Journal

LANGUAGE: Russian

AB New shuttle vectors (F-60 and F-61) encoding chloramphenicol acetyltransferase (CAT) antisense RNA under the control of multiple cauliflower mosaic virus 35S promoter enhancer elements, when introduced into tobacco via Agrobacterium-mediated leaf disk transformation, inhibited CAT activity of transgenic plants with a genome integrated CAT gene. Similar inhibition of CAT expression was observed in tobacco transformed by a shuttle vector encoding CAT antisense RNA under the control of the Cab promoter from the wheat chlorophyll a/b-binding protein gene.